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# The Relationship Between Liver Fibrosis Scores and Ascending Aortic Dilatation

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## ABSTRACT

**Background & Aims**: Non-alcoholic fatty liver disease (NAFLD) is related to an increased atherosclerotic cardiovascular disease (ASCVD) risk. This study investigated a potential relationship between liver fibrosis scores (LFS) reflecting NAFLD and ascending aortic dilatation (AAD)

**Methods**: This is an observational and cross-sectional study. Patients were consecutively enrolled from a cardiology clinic. The NAFLD fibrosis score (NFS), fibrosis-4 (FIB-4) index, aspartate aminotransferase (AST) to platelet ratio (APRI), and BARD scores of each patient were calculated. The ascending aortic diameters were evaluated by transthoracic echocardiography according to current clinical guidelines. The patients were allocated into two groups with and without AAD.

**Results**: A total of 272 patients were included in the study. In AAD group, age, patients with hypertension (HT), coronary artery disease (CAD), FIB-4 index, BARD score and the NFS were significantly higher. As compared to the AAD group, body mass index (BMI), hemoglobin, and diuretic use were significantly higher in patients without aortic dilatation. The NFS with AAD, and NFS and FIB-4 index with indexed aortic diameter (AI) showed significant positive correlation (R=0.546, R=0.332, R=0.314 with p<0.001, respectively). In multivariate logistic regression analysis hemoglobin levels (OR=0.728, 95%CI: 0.553-0.958; p=0.023), BMI (OR=0.762, 95%CI: 0.668-0.869, p<0.001), HT (OR=3.269, 95%CI: 1.045-10.220; p=0.042), BARD score (OR=1.248, 95%CIL 0.815-1.955; p=0.044), and FIB-4 index (OR=2.432, 95%CI: 1.395-4.246; p=0.002) were found to be independently related to AAD.

**Conclusions**: Our study demonstrated a statistically significant relationship between NFS, FIB-4 index, BARD score and AAD. The presence of positive correlation among LFS and AAD in our study is remarkable. This may emphasize the increased risk of AAD in NAFLD.

**Key words:** non-alcoholic fatty liver disease – NAFLD – fibrosis – liver fibrosis scores – aortic dilatation – FIB4 – APRI – NFS – BARD.

**Abbreviations**: AAD: ascending aortic dilatation; AI: aortic diameter index; ALT: alanine aminotransferase; APRI: aspartate aminotransferase to platelets ratio index; AST: aspartate aminotransferase; BMI: body mass index; CI: confidence interval; CVD: cardiovascular disease; DM: diabetes mellitus; FIB-4: fibrosis-4 index; HDL: high-density lipoprotein; HT: hypertension; LDL: low density lipoprotein; LFS: liver fibrosis scores; LVH: left ventricular hypertrophy; NAFLD: non-alcoholic fatty liver disease; NFS: NAFLD fibrosis score; OR: odds ratio; TG: triglycerides; TTE: transthoracic echocardiography.

## INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is among the most prevalent metabolic liver disorders with a prevalence up to 30% in developed countries. The incidence of NAFLD is expected to increase exponentially in the near future because of an increased number of elderly individuals, sedentary lifestyle, and obesity [1].

Cardiovascular disease (CVD) is significantly related to the degree of liver fibrosis and is the leading cause of death in patients with NAFLD [2, 3]. This close relationship between NAFLD and CVD is due to the liver's central role in glucose and lipid metabolism, independent of traditional cardiometabolic risk factors such as diabetes mellitus (DM), obesity, hypertension (HT), and dyslipidemia [4].

Age dependent dilatation of aorta is related to underlying alterations in the aortic wall including calcification, decreased elastin component, accumulation of collagen and elastin breaks due to long term exposure of traditional risk factors such as HT, metabolic syndrome, hyperuricemia, and smoking [5]. Dilated aortic root was reported to be a sign of organ damage in parallel with other subclinical markers which have intrinsic prognostic value such as left ventricular hypertrophy (LVH), carotid atherosclerosis, diminished glomerular filtration rate, microalbuminuria, and increased pulse wave velocity [6]. Inflammation is a physiologic reaction induced by fibrosis that facilitates the healing of tissue damage. However, several factors such as monocyte/macrophages can induce an exaggerated inflammatory response and pathologic remodeling may occur because of the impaired healing fibrosis process. Similar mechanisms can be observed in aortic aneurysm formation [7].

Liver biopsy has been the golden standard to define histopathologic characteristics in NAFLD, but it cannot be performed in every patient because of cost and complications. For this reason, researchers have developed several non-invasive models to supplant liver biopsy [8, 9]. The NAFLD fibrosis score (NFS), fibrosis-4 index (FIB-4), aspartate aminotransferase to platelets ratio index (APRI) and BARD score are formulations developed to exclude severe hepatic fibrosis and their role in initial diagnosis is incontrovertible [10].

Considering the shared pathophysiological mechanisms between aortic dilatation and NAFLD, we hypothesized a significant association between these two conditions. Moreover, the recognized link between NAFLD and CVD suggested a potential relationship between liver fibrosis scores (LFS) reflecting NAFLD and ascending aortic dilatation (AAD).

## METHODS

### Study Population

This wass a single center, prospective, cross sectional, and observational study. Patients admitted to cardiology outpatient clinic for routine checkup examination who met the inclusion criteria were consecutively enrolled in our study. All patients were evaluated by transthoracic echocardiography (TTE) to rule out AAD. Routine blood analysis from each patient was performed to calculate NFS, FIB-4 index, BARD score, and APRI. The basic demographic characteristics, biochemical and relevant clinical data of each patient were recorded. The patients with acute or chronic kidney failure, active inflammatory disease, heart failure, valvular heart disease or valve prosthesis, endocrinologic disorder, electrolyte imbalance, anemia, pulmonary embolism, malignancy, cirrhosis, or chronic hepatic failure due to a known etiology (viral hepatitis, hemochromatosis, alcohol abuse) were excluded.

The local institutional Ethics Committee approved the study (The Ethics Committee decision date and decision number is: 03/08/2023, E-40465587-050.01.04-762). All patients were informed, and written consents were taken for the study.

### Laboratory Analyses

Blood samples for routine tests were collected from the brachial vein by venous puncture after a minimum of 8 hours of fasting. Liver serological markers were studied for the diagnosis of acute and chronic hepatitis. Serum fasting glucose, creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), albumin and lipid levels including low density lipoprotein (LDL), triglycerides (TG) and high-density lipoprotein (HDL), were measured by standard techniques. White blood cell (WBC, leucocyte), thrombocytes (PLT, platelet) numbers were derived from automated cell counter (Coulter Gen-S, COULTER Corp, Miami, USA). Body mass index (BMI) was calculated as the weight divided by the square of height in meters.

### Non-invasive Liver Fibrosis Scores

The NFS, FIB-4 index, APRI, and BARD scores were utilized to categorize patients with NAFLD whether they had a risk of advanced liver fibrosis or not. NFS was calculated by the formula; -1.675 + (0.037*age [years]) + (0.094*BMI [kg/m$^2$]) + (1.13*IFG/DM [yes = 1, no = 0]) + (0.99*AST/ALT ratio) – (0.013*platelet count [×10$^9$/L]) – (0.66*albumin [g/dl]). FIB-4 index was calculated by the formula; [Age (year) × AST] / [platelets × √(ALT)]. APRI was calculated by the formula; (AST in IU/L) / (AST Upper Limit of Normal in IU/L) / (Platelets in 10$^9$/L). BARD score was calculated by the addition of the selected points: BMI ≥28 no=0, yes=1; AST/ALT ratio ≥0.8 no=0, yes=2; DM no=0, yes=1. A practical automated online calculator was used (https:// https://www.mdcalc.com/) [9]. NFS < -1.5 indicate low, scores between > -1.5 to < 0.67 indicate intermediate, and scores > 0.67 indicate high probability of liver fibrosis [8-10].

### Transthoracic Echocardiography

All patients were evaluated by 2D TTE by two licensed sonographers as recommended by clinical practice guidelines [11]. The measurements were made by using 1-5 MHz X5-1 transducer probe and iE33 echocardiography machine (Philips Epiq 7 systems, Philips Medical Systems, Andover, MA). The thoracic aorta from the aortic annulus to the innominate artery was imaged; the diameters of aortic annulus, sinus of Valsalva, sinotubular junction and proximal tubular ascending aorta (AA) were measured. The tubular AA diameters were measured 3 cm above the aortic valve. The echocardiographic images were obtained from standard parasternal long axis window and the measurements were indexed to the body surface area [11, 12]. The aortic diameters were measured at end diastole from inner-to-inner edge to increase reproducibility. To decrease the overestimation of the sizes, the aorta was measured perpendicular to its long axis to prevent an oblique image plane. Aortic diameter index (AI) >21 mm/m$^2$ was defined as aortic dilatation. All images were stored digitally for offline analysis (QLAB 10.0 cardiac 3DQ, Philips Medical Systems).

### Statistical Analysis

Visual (histograms, probability graphics) and analytic (Kolmogorov–Smirnov/Shapiro–Wilk test) methods were used to determine normal distribution of variables. To test homogeneity of variances, the Levene test was performed. The continuous and categorical variables were presented as mean±standart deviation and percentages, respectively. The variables with non-normal distribution were presented

as median and interquartile range. To compare categorical groups, Chi-square, or Fisher's exact test (when Chi-square test hypothesis fails due to expected low cell numbers) was used. Normally distributed parameters were assessed by the two tailed student t-test and the Mann-Whitney U test was used for continuous variables without normal distribution. Univariate and backward multivariate logistic regression analysis was performed. To determine the related independent variables for AAD, multivariate logistic regression analysis was used. The results were presented as 95% confidence interval (CI) and odds ratio (OR). Two-sided p value < 0.05 was assumed to be statistically significant. The statistical analyses were performed by SPSS software 21.0 statistics package (SPSS IBM. Inc.).

## RESULTS

A total of consecutive 272 patients without significant history of alcohol use who were admitted to the cardiology clinic for a routine cardiology checkup were included our study. The flow diagram illustrates the patient recruitment process for the study (Fig. 1). 116 women and 156 men with mean age of 54.1±13.8 were included in the analysis. 34 (12.5%) patients had AAD. The demographic and laboratory data of the patients with and without AAD were compared (Table I). In the group with AAD, age, patients with hyperlipidemia (HL), HT and coronary artery disease, and NFS, FIB-4 index, and BARD scores were significantly higher, but hemoglobin levels and BMI were lower. Remarkably, APRI did not show a statistically significant difference between the groups in contrast to other LFS. Additionally, without reaching statistical significance, the number of patients using angiotensin converting enzyme inhibitor (ACEi) and diabetics were higher; the number of patients using oral antidiabetic drug (OAD)/insulin treatment and number of males, and serum AST and albumin levels were lower compared to the group without AAD.

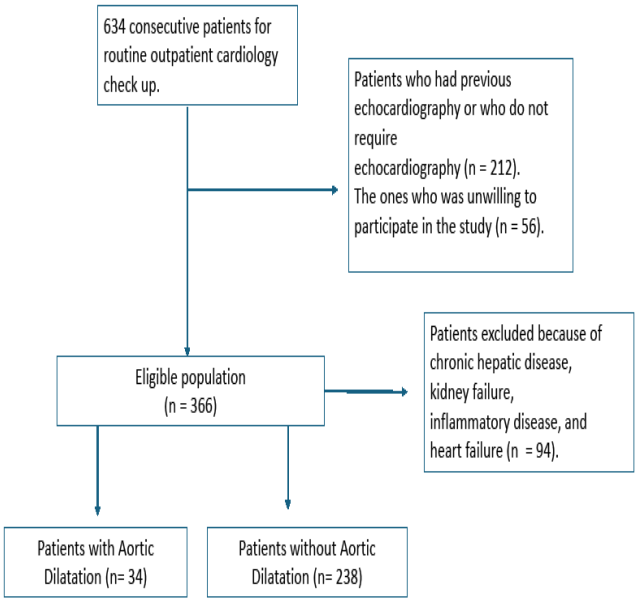


**Fig. 1.** The flow diagram illustrates the patient recruitment process for the study.

**Table I.** The demographic and laboratory data of the patients

| Variable | Ascending Aortic Dilatation (-) (n=238) | Ascending Aortic Dilatation (+) (n=34) | p |
|---|---|---|---|
| Demographic Data | | | |
| Age (years) | 53.4±13.4 | 62.6±11.5 | <0.001 |
| Gender (Male), n (%) | 141 (59.2) | 15 (44.1) | 0.070 |
| BMI | 29.5±4.8 | 26.4±3.9 | <0.001 |
| HL, n (%) | 56 (23.5) | 15 (44.1) | 0.011 |
| DM, n (%) | 44 (18.6) | 12 (34.3) | 0.043 |
| HT, n (%) | 101 (42.4) | 22 (64.7) | 0.012 |
| CAD, n (%) | 104 (43.7) | 22 (64.7) | 0.017 |
| Current Smoking, n (%) | 96 (40.7) | 15 (44.1) | 0.420 |
| Beta Blocker, n (%) | 49 (20.6) | 10 (29.4) | 0.171 |
| ACE-i, n (%) | 48 (20.2) | 11 (32.4) | 0.086 |
| ASA, n (%) | 69 (29) | 14 (41.2) | 0.108 |
| ARB, n (%) | 39 (16.4) | 6 (17.6) | 0.508 |
| Statin, n (%) | 34 (14.3) | 4 (11.8) | 0.467 |
| CCB, n (%) | 31 (13) | 5 (14.7) | 0.480 |
| OAD/Insulin, n (%) | 44 (18.5) | 11 (32.4) | 0.054 |
| Laboratory Data | | | |
| Glucose (mg/dL) | 112.3±42.2 | 123±41.4 | 0.147 |
| WBC ($10^3$/μL) | 8.2±2.3 | 8.1±2.5 | 0.568 |
| Neutrophil ($10^3$/μL) | 5.2±2.1 | 5.4±2.4 | 0.599 |
| PLT ($10^3$/μL) | 249±66 | 247±84 | 0.887 |
| CRP (mg/dL)* | 4.25 (2.6-6.5) | 3.8 (2.5-6.3) | 0.643 |
| Hemoglobin (g/dL) | 14±1.7 | 12.9±1.7 | 0.001 |
| Serum Creatinine (mg/dL) | 0.85±0.24 | 0.91±0.27 | 0.228 |
| AST (mg/dL) | 24.5±11.3 | 28.4±25 | 0.130 |
| ALT (mg/dL) | 23.2±13.9 | 19.1±7.9 | 0.092 |
| Albumin (gr/dL) | 4.2±0.51 | 4.07±0.35 | 0.094 |
| Cholesterol (mg/dL) | 206±49.5 | 219±51.7 | 0.170 |
| HDL (mg/dL) | 48.1±12.1 | 50.1±12.1 | 0.334 |
| LDL (mg/dL) | 132±38.9 | 140±43 | 0.259 |
| TG (mg/dL) | 149.8±83 | 154.1±77.1 | 0.728 |
| NFS | -2.5±2.2 | -1.5±2.4 | 0.020 |
| FIB-4 | 1.26±0.94 | 1.97±1.91 | <0.001 |
| APRI | 0.22(0.16-0.31) | 0.20(0.16-0.34) | 0.845 |
| BARD score | 1.89±1,01 | 2,28±1,04 | 0,035 |

ACEi: angiotensin converting enzyme inhibitor; ALT: alanine amino transferase; APRI: aspartate amino transferase to platelet ratio index; ARB: angiotensin receptor blocker; ASA: acetyl salicylic acid; AST: aspartate amino transferase, BMI: body mass index; CAD: coronary artery disease; CCB: calcium channel blocker; CRP: C reactive protein; DM: diabetes mellitus; FIB-4: fibrosis-4 index; HDL: high density lipoprotein; HL: hyperlipidemia; HT: hypertension; LDL: low density lipoprotein; NFS: non-alcoholic fatty liver disease fibrosis score; PLT: platelet; TG: triglycerides; WBC: white blood cell. Continuous variables are given as mean ± SD. *: median, interquartile range (range, [25% percentile-75% percentile])

The correlation analyses of NFS with AAD and AI, and FIB-4 index with AI were performed. In correlation analysis of NFS with AAD and AI, and FIB-4 index with AI, the results were

R=0.546, P<0.001; R=0.332, P<0.001; and R=0.314, P<0.001; respectively (Figures 2-3, and 4, respectively).

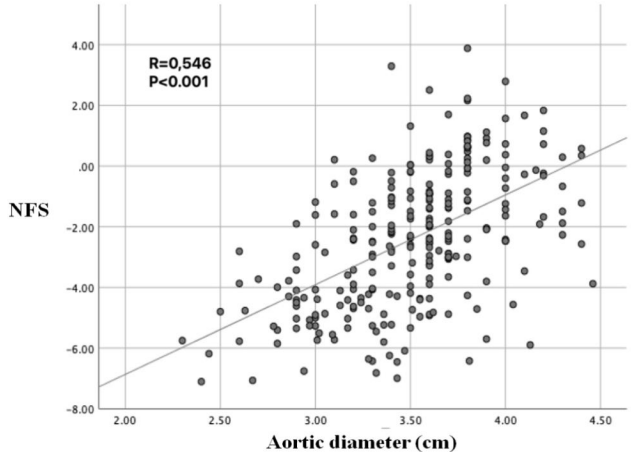


**Fig. 2.** The simple scatter graphic with fit line of NAFLD fibrosis score (NFS) by aortic diameter (AD).

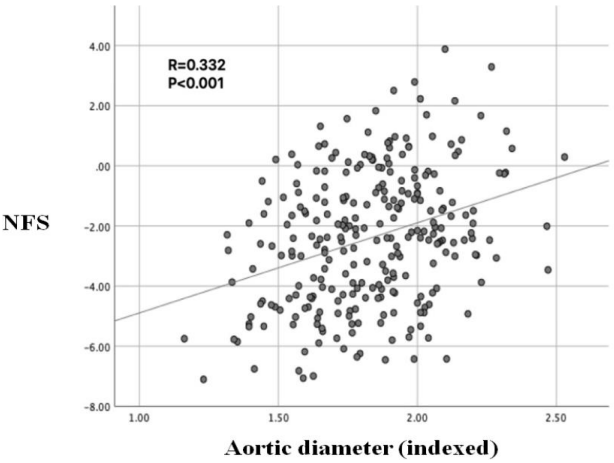


**Fig. 3.** The simple scatter graphic with fit line of NAFLD fibrosis score (NFS) by indexed aortic diameter (AI).

The parameters which had statistically significant difference between the two groups were analyzed by univariate and multivariate logistic regression analysis (backward method), respectively (Table II). Covariable factors potentially influencing NAFLD were excluded from the regression analysis. To address this, two models were constructed. In model 1, we incorporated all variables except FIB-4 index and BARD score. In model 2, we incorporated all variables in multivariate (backward method) logistic regression analysis. Hemoglobin levels (OR=0.710, 95%CI: 0.559-0.903; p=0.005), age (OR=1.040, 95%CI: 0.998-1.085; p=0.064), BMI (OR=0.791, 95%CI: 0.709-0.882; p<0.001), and NFS (OR:1.422, 95%CI: 1.176-1.716; p<0.001) were found to be independently related to AAD in model 1. Hemoglobin levels (OR=0.728, 95%CI: 0.553-0.958; p=0.023), BMI (OR=0.762, 95%CI: 0.668-0.869, p<0.001), HT (OR=3.269, 95%CI: 1.045-10.220; p=0.042), BARD score (OR=1.248, 95%CI: 0.815-1.955; p=0.044), and FIB-4 index (OR=2.432, 95%CI: 1.395-4.246; p=0.002) were found to be independently related to AAD in model 2. In ROC analysis, NFS had an AUC value of 0.694 with p<0.001 and FIB-4 index had an AUC value of 0.679 with p<0.001 (Figs. 5 and 6).

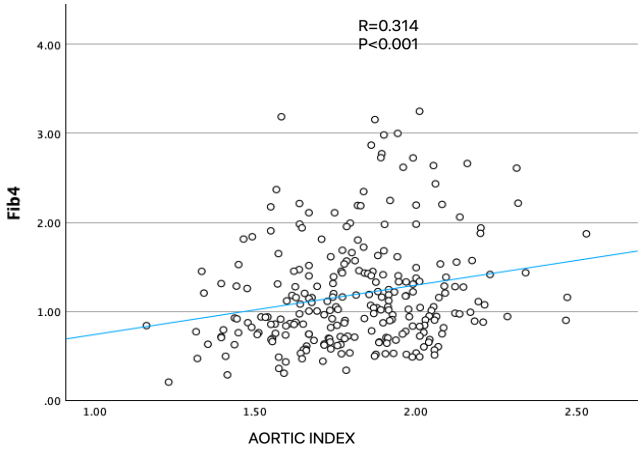


**Fig. 4.** FIB-4 and aortic index correlation graph.

## DISCUSSION

Since NAFLD is a common disease in the community, and has a close relationship with CVD, it is important to investigate the relationship between NAFLD and aortic health to take preventive measures. This study revealed several important

**Table II.** The univariate and multivariate (model 1 and 2) logistic regression analysis of the variables

| | Univariate | | | Multivariate (Model 1) | | | Multivariate (Model 2) | | |
|---|---|---|---|---|---|---|---|---|---|
| Variable | OR | 95% CI | p | OR | 95% CI | p | OR | 95% CI | p |
| Age | 1.058 | 1.026-1.091 | <0.001 | 1.040 | 0.998-1.085 | 0.064 | | | |
| BMI | 0.863 | 0.793-0.938 | 0.001 | 0.791 | 0.709-0.882 | <0.001 | 0.762 | 0.668-0.869 | <0.001 |
| HT | 2.487 | 1.176-5.259 | 0.017 | | | | 3.269 | 1.045-10.220 | 0.042 |
| HL | 2.424 | 1.164-5.048 | 0.018 | | | | | | |
| CAD | 2.362 | 1.117-4.994 | 0.024 | | | | | | |
| Hemoglobin | 0.739 | 0.604-0.904 | 0.003 | 0.710 | 0.559-0.903 | 0.005 | 0.728 | 0.553-0.958 | 0.023 |
| NFS | 1.212 | 1.028-1.428 | 0.022 | 1.338 | 1.016-1.761 | 0.038 | | | |
| FIB-4 | 1.615 | 1.212-2.154 | 0.001 | | | | 2.432 | 1.395-4.246 | 0.002 |
| BARD | 1.131 | 1.001-1.318 | 0.042 | | | | 1.248 | 0.815-1.955 | 0.044 |

For abbreviations see Table I.

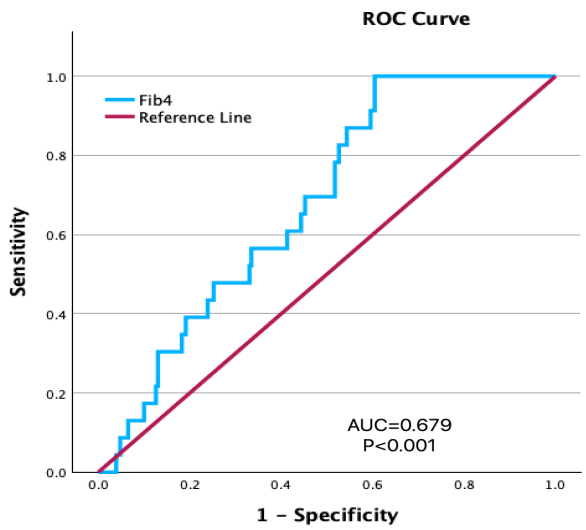


**Fig. 5.** ROC curve for FIB-4 index.

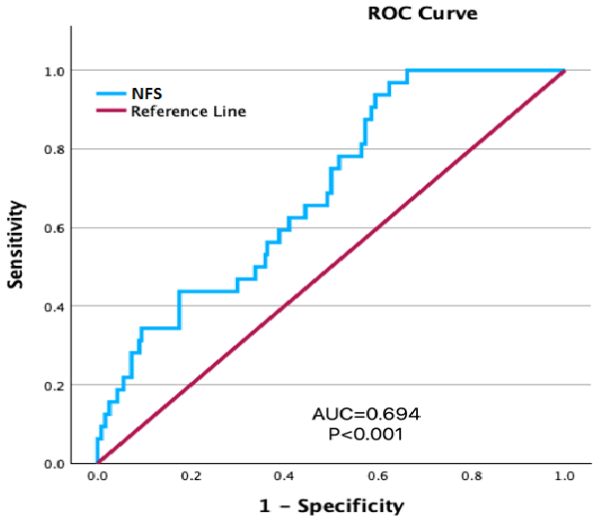


**Fig. 6**. ROC curve for NAFLD fibrosis score (NFS).

findings. Initially, LFS, including NFS, FIB-4 index, and BARD score, were identified as independent predictors of AAD. Additionally, there was a positive correlation between LFS, particularly NFS and FIB-4 index, and ascending aorta diameter. To the best of our knowledge, this is one of the first studies on this subject and will shed light on future studies in terms of elucidating the relationship between LFS, which indicate NAFLD, and AAD. This result may emphasize the clinical importance of LFS and may suggest a strategy with a closer follow up of the patients for AAD in long term. In addition, low hemoglobin level, advanced age, HT, low BMI were found to be independent predictors of AAD in our study.

Ascending aortic dilatation is a life-threatening insidious condition in which early diagnosis can be barely done before a catastrophic rupture. Most of the time, dilatation of the thoracic aorta is detected incidentally during routine imaging. The true incidence of AAD including the undiagnosed ones in the general population has not been studied in detail. However, the prevalence of AAD was shown to be 2,8 % in individuals 50 years of age and older if the cut off for AAD defined is ≥4,0 cm [13]. Also, the total rate of incidentally detected thoracic aorta aneurysms (TAA) was reported to be 7.6 in 100000 [14]. Although there is not any epidemiological study, according to our observations, AAD is a common finding in the region where our study patients live. In our study, 34 out of randomly selected 272 patients who admitted to our outpatient clinic had AAD with a relatively higher rate of 12.5%.

The association of advanced age and HT with AAD has been well documented in previous studies. Our results are consistent with the literature in this context. AAD has a close relationship with increased cardiovascular events [15]. In the study by Rogers et al. [16], the association between aortic diameters and traditional risk factors were evaluated and aortic diameters had significant correlation with age and BSA in age adjusted analysis. Additionally, they concluded that CVD risk factors such as diastolic blood pressure and smoking had a significant positive correlation with aortic diameters [16]. Eswarsingh et al. [17] reported that there was a significant association between the aortic root and AA dilatation and risk factors such as age, gender, and HT. However, there was no significant relationship between the dilatation progression rate and BMI, DM, smoking and HL in the 10 years follow up [17]. It was previously reported that smooth muscle cell loss and degradation of elastic fibers due to ageing had some role in AAD together with several complex mechanisms [18]. Although smoking was associated with AAD and dissection in several studies [19], there was no significant relationship between smoking and AAD in our study in parallel with the findings of Eswarsingh et al. [17]. In the study by Landenhed et al. [19], they did not observe significant relationship with obesity in any aortic disease. However, another study showed significant association with obesity and abdominal aortic aneurysms [20].

We observed that NFS and FIB-4 index, which include the age component in their formula, showed statistical significance. Similarly, the analysis of the BARD score, which does not include an age component in its formula, also showed statistical significance. However, the APRI score without an age component did not show statistical significance. As previously noted, while previous studies have established a correlation between AAD and aging, our study revealed a significant association between AAD and LFS with and without age as a multiplier in their formula. Therefore, according to these results, it may be considerable to focus on the potential association between NAFLD and AAD independent of age.

Interestingly, BMI was lower in patients with AAD in our study. This can be explained by a lower number of individuals in our study or the suboptimal strength of BMI in determination of degree obesity and should be discussed in further studies [21]. In addition, although it is known that the incidence of NAFLD increases with obesity, a significant number of patients with NAFLD are lean individuals. In a meta-analysis by Ye et al. [22] it was shown that 40% of NAFLD patients were non-obese or lean individuals.

Remarkably, low hemoglobin level was found to be strongly associated with AAD in our study. Previous studies have shown a significant relationship between low hematocrit level and vascular functions. The oxygen delivery mechanisms at various

levels of hemoglobin and hematocrit might be associated with impaired vascular function [23]. Persistent hypoxia can impact endothelial function by elevating inflammation and oxidative stress levels [24]. This finding needs to be supported by larger studies.

The inflammation has complex role at the beginning and progression of aortic disease including AAD and acute aortic syndromes that necessitates further studies to elucidate pathophysiologic mechanisms underlying aortic aneurysms [17-20, 25]. Adventitial fibroblasts are one of the basic components of the aortic wall and have crucial role in production of extracellular matrix, protection of the structure and function of the aorta. These fibroblasts are the important mediators of inflammation and if they are activated by any stress or injury, they can transform into myofibroblasts, migrate to the injury site, and produce collagen and cytokines and results in undesirable remodeling if this process becomes persistent [26].

The mechanisms that link NAFLD to CVD are complex and contain so many different pathways. These two share several risk factors and both are the result of end-organ damage due to metabolic syndrome. The processes in NAFLD such as increased systemic inflammation, changes in vascular tone, promotion of plaque formation, insulin resistance, dyslipidemia, oxidative stress, platelet activation and endothelial dysfunction were proposed to increase risk of CVD [27]. There are also several biologic processes previously proposed by Mikołajczyk et al. [28] such as perivascular fibrosis, remodeling of the vascular wall, elastin loss, inflammation, oxidative stress, and apoptosis of vascular cells which contribute to the progression of an aortic aneurysm. In the study of Quintana et al. [29], fibrosis at the cellular level was reported to promote development of aortic aneurysm. Our findings seem to be consistent with the current scientific data that AAD may be seen in patients with severe fibrosis risk with higher LFS.

### Study Limitations

This study is a single center study with a relatively lower sample size. The study population includes individuals without a significant history of alcohol consumption who were admitted to our clinic for routine cardiology check-ups. The patients consisted of middle-aged local people living nearby. So, the findings of our study may not be generalized to different age and ethnic groups. Since, the data was collected from a cross-sectional study; we are not able to make a causation regarding our results. In our study, we did not use any imaging method for the diagnosis of NAFLD. More comprehensive results can be obtained by including different imaging methods in the study. Another limitation of the study is the measurement of aortic diameters by transthoracic echocardiography only without confirmation of either computed tomography or magnetic resonance imaging.

## CONCLUSIONS

The diagnosis of AAD and determination of its risk factors is staminal. Because most of the aortic aneurysms are asymptomatic, they generally are detected incidentally during imaging for other etiologies. In this cross-sectional study, AAD was observed to be higher in patients with higher LFS including NFS, FIB-4 index and BARD score. This finding may show the importance of fibrosis and inflammation in the pathogenesis of AAD. We recommend that physicians be encouraged to screen aortic diameters in patients with NAFLD.

**Conflicts of interest**: None to declare.

**Authors' contributions**: Hakan Duman conceived and designed the study. Hakan Duman, Handan Duman and A.S. collected and assamblied the data. Handan Duman analyzed the data. Hakan Duman, Hüseyin Durak and E.I. wrote the manuscript. Hüseyin Durak and E.I. criticaly revised the manuscript. Hakan Duman, Handan Duman and A.S. approved the final version of the manuscript. All authors have read and agreed to the published version of the manuscript.

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